WC 0400

Structural and Functional Management of the Diabetic Foot

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At some time in their career, all primary care, family practice, and podiatric physicians will encounter diabetic patients. Treating wounds and chronic ulcerations in the diabetic patient is challenging, frustrating and often times very disappointing for all concerned. The diabetic patient is plagued with many contributing factors that lead to multifaceted complications. The diabetic patient presents to the physician a very involved group of conditions that lead to breakdown of the

EXHIBIT "D"

Figure 1. First metatarsal head resected removing all of the osteomyelitic bone. The shaft is cut on an angle to decrease the possibility of any bony prominence medially. A new increase in peak pressure and shear force now exist on the second metatarsal head.

Figure 2. Third metatarsal head resected increasing the peak pressure and the shear forces on the adjacent metatarsal heads.

Figure 3. Pan–metatarsal head resections of all metatarsals. A new parabola is created, allowing equal weight bearing across the transverse surface of the forefoot.

skin and deeper tissues often creating loss of function. Neuropathy, in combination with angiopathy, significantly challenges the clinician attempting to heal wounds of the diabetic foot.

Neuropathy is perhaps the most dangerous complication, as it cuts off the primary communication between the lower extremity and the central nervous system. The patient gets no signals from the insensate foot and consequently goes through denial regarding the impending seriousness of his or her condition. This denial should be considered another complication of diabetes; it is a very real entity that the treating physician must take into account when treating this category of patient.

The treatment of diabetic wounds is complex because even when properly managed, the wounds may not heal as well as expected, and when they do, the closure is often temporary and difficult to maintain.

The care of the diabetic is best managed by a team of experts familiar with long term patterns of ulceration and wound healing. This article will attempt to provide an orderly approach to the structural and functional management of the diabetic patient.

The Forces

Friction is created by one body moving over another body. The more irregular the metatarsal distribution or bone shape, the greater the friction, thus resulting in tissue breakdown. Continual shoe friction against the skin forms blisters. Continual friction on the weight-bearing or dorsal surface of the foot may cause callouses or corns.

Pressure is developed by a force created by weight against another body part. As the area of the surface increases, the pressure to that area decreases.[1,4,5] In a particular bony prominence where the area is small, as in a bone spur, the pressure per unit area of skin increases. The result of the increase of pressure in the diabetic foot will lead to tissue breakdown secondary to ischemia.

Shear is developed during the gait cycle. This is not as apparent as friction or pressure but the effect is much greater and often overlooked when treating the patient. It is quite insidious and must be controlled and prevented in order to reduce present and future ulcer development. The shear force develops when one bone sliding within the deep tissue causes a combination of friction and pressure to be exerted on the adjacent tissue. This force is perpendicular to the plane of the supporting surface and functions in a rotary direction.

Distribution of Force

At heel contact, during the gait cycle, the heel bears weight for approximately 55 to 65% of the stance phase, and reaches its peak force of 70to 80% of body weight at approximately 15 to 20% into the stance phase. The most posterior and plantar aspect of the calcaneus receives the greatest pressure per time segment.[1,4,5]

The metatarsal heads bear weight for approximately 80% of the gait cycle, and the peak pressure is present towards the last 25% of the gait cycle. The first metatarsal head bears weight after the fifth metatarsal head contacts the ground, and pressure under the first metatarsal head ends later than the fifth metatarsal head. The amount of peak pressure among the metatarsals is highly variable. Maximum peak pressure is sometimes found under the first metatarsal head or under the second metatarsal head. This is dependent upon the length of the individual metatarsal.

Mid-foot pressure is variable, but usually low when compared to forefoot pressures. The average contact time for the midfoot is similar to that of the heel, about 55 to 60% of the stance phase.[1-7]

Shear markedly decreases the high peak pressure seen at the posterior of the heel. It also serves to decrease the peak pressure of the lateral three metatarsal heads so that the first metatarsal head reaches the greatest peak pressure of all the metatarsal heads.

In diabetics, peak forces have been noted on the medial heel and lateral three metatarsal heads as well as the first and second metatarsal heads. Ulcers invariably occur at the sight of maximum peak force in each foot.

The first metatarsal head is the most common area for breakdown.[8,9] In a normal pattern of motion, the foot will have gradual heel contact, then a gentle rolling motion. This pronation allows a gradual shifting of weight from one bone to another.

The actual planning of the procedure is an important step in treating a patient presenting with osteomyelitis of the first metatarsal head. Understanding that the first metatarsal head bears the majority of weight at propulsion, a resection of the metatarsal will consequently transfer all of the weight to the adjacent metatarsals. This will disrupt the normal progression of weight bearing shift. A logical staged sequence needs to be carried out when metatarsal resections are planned.

Stage I. The infected metatarsal is resected thus decreasing the peak pressure which was the initiating cause of the ulcer. The metatarsal head is resected and the metatarsal shaft is resected as far proximally as needed to eradicate any osteomyelitic bone. The ulcer is surgically excised, removing all of the necrotic and fibrinous material. A clean, firm granulating bed should be present. This wound is packed open with sterile saline gauze. The patient is placed on appropriate antibiotics determined by bone cultures.

Stage II. The patient remains 100% non–weight bearing. Saline dressings wet to dry are changed twice a day. The dressings should be placed deep into the excised wound cavity. Office debridements, removing any fibrin and necrotic tissue, continue.

Stage III. When the plantar wound has completely closed, the forefoot must be surgically rebalanced. The functional and structural integrity of the forefoot is paramount in reducing the risk of unwanted future peak pressure and shear forces yielding new ulcerations. The forefoot biomechanical forces are redistributed equally. The weight bearing surface is spread out transversely over all five metatarsal shafts. The removal of one metatarsal increases the peak pressures and shear forces on adjacent metatarsals. To ensure equal distribution of weight, rebalancing of peak and shear forces are performed by resecting the remaining metatarsals heads. By removing the appropriate adjacent metatarsal heads the distal parabola of metatarsals are restored. The peak pressures are evenly distributed to all the osteotomized ends of the metatarsals. With equal pressure the risk of transfer ulcerations is diminished. (See Figures 1, 2, and 3.)

Stage IV. Once the forefoot is structurally balanced and able to function with equal weight distribution, orthoses can easily be constructed and utilized. A triple laminate orthotic device, constructed from a mold of the foot, not only accommodates the foot by cushioning abnormal bony prominences, but also supports the foot in a neutral position, allowing proper foot function throughout the gait cycle.

References

1. Roy KJ: Force, Pressure and motion measurements of the foot. *Clin Pod Med Surg* 1988;5:491–508.
2. Elftman H: A cinematic study of the distribution of pressure in the human foot. *Anat Rec* 1934;59:481–491.
3. Soames PW: Foot pressure patterns during gait. *J Biomed Eng* 1985; 7:120–126.
4. Lord M: Foot pressure measurement. *J Biomed Eng* 1981; 3:91–99.
5. Tappin JW: Method of measuring forces on the sole of the foot. *Clin Phys Physiol Meas* 1980;1:83–85.
6. Stott JRR: Forces under the foot. *JBJ* 1973;55B:335–344.
7. Pollard JP: Forces under the foot. *J Biomed Eng* 1983;5:37–40.
8. Sanders LJ: Amputation in the diabetic foot. *Clin Pod Med Surg* 1987;4:481-501.
9. Daniels EG: Prevention of complications of partial foot amputations. *Clin Pod Med Surg* 1987;4:503-501.

PREFACE

Virtually every primary care physician treats diabetic patients, and nearly half of these patients have—or ultimately will have—diabetic neuropathy.

This most common disabling complication of diabetes suppresses the warning signal of pain that helps patients avoid injury or alerts them to take action when it occurs.

Since the human race became erect, the human foot has been a prime target for injury and it is all but inevitable that patients with unprotected insensate feet will eventually become "walking wounded."

Once these wounds occur, they are both dangerous and frustrating. Stubbornly resistant to treatment, they tend to heal poorly, if at all, and are prone to recurrence. Moreover, the non-healing ulcer is frequently complicated by polymicrobial infections, leading to osteomyelitis, gangrene, and the ultimate insult of amputation.

This brief overview of neuropathic ulcers and the diabetic foot is not intended as a substitute for the volumes that have been written on the subject. Instead, it is designed to provide practical tools to assist the primary care physician—who will usually be the first to confront the problems—in assessing and caring for the feet of their diabetic patients.

Specifically, it addresses:

- How to recognize and quantify diabetic neuropathy as it pertains to feet and lower extremities.
- How to minimize injury and ulceration in susceptible patients.
- How to determine which wounds are amenable to treatment and how they are best approached.
- When it is preferable to refer care to a comprehensive wound management team.
- What state-of-the-art management can provide to help heal recalcitrant wounds and prevent amputation.
- How to monitor and manage healed wounds to prevent recurrence.

The burden on diabetic feet

Of the approximately 14,000,000 people with diabetes in America today, some 2,000,000 (15%) may develop foot or leg ulcers during their lifetime.[1] As a result, a potential 50,000 will undergo major amputations[2] at an average cost of $25,000.[3]

Moreover, statistics show that roughly half of these amputees will develop a limb threatening condition of the contralateral limb within 18 months, and over half will require a contralateral amputation within 3 to 5 years if they are among the 35-40% who survive long enough.[4]

While diabetes itself is the fundamental problem, it is diabetic neuropathy that places the diabetic foot at such extreme risk. Since the discovery of insulin, medical advances have prolonged survival for people with diabetes so that, in America today, 90% are over 40 years of age and millions are well into their 60s.[5] Diabetic neuropathy increases with age and duration, and after 20 years of the disease, some 42% of people with Type I diabetes will exhibit it.[6] The incidence may be somewhat lower with Type II disease, but the prevalence is greater.

Without pain as a warning, trivial injuries can become threats to limbs—and even life—long before the patient complains. Yet evaluation of the diabetic patient's feet by their physician or health professional often does not occur. In one survey of physician's practices, only 12.3% indicated that they routinely examined their patients' feet.[7]

The critical factor is that most diabetic patients with neuropathy aren't aware of what's happening to their feet and thus, their physician is the primary person to assist them in reducing their risk of ulcers and/or amputation.

Diabetic neuropathy

Diabetes causes both angiopathy and neuropathy, and both contribute to the special vulnerability of the diabetic's feet to injury. Approximately three to five times as many patients are admitted as a result of painless foot trauma as for ischemic pain.[8] **Primary** ischemic ulcers are relatively rare.

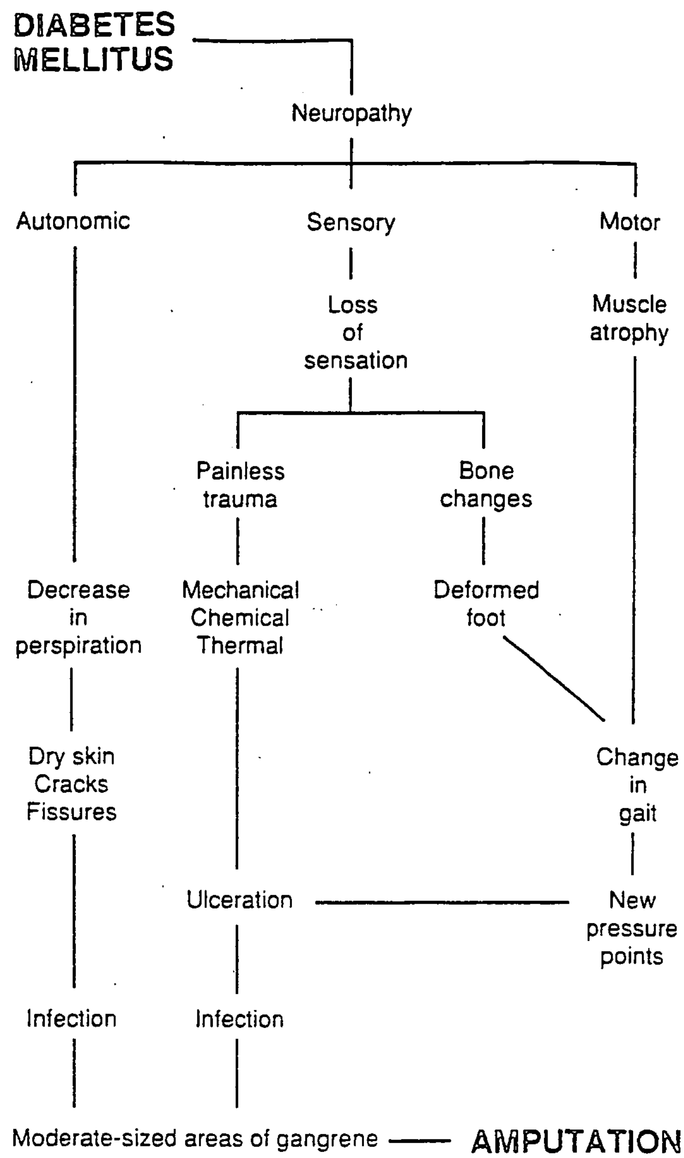
Peripheral neuropathy develops gradually and insidiously in both Type I and Type II people with diabetes.

The precise etiology of diabetic neuropathy remains unclear, but both vascular and metabolic factors are implicated. It parallels both the duration and severity of hyperglycemia, and the most important lesion is loss of myelinated and unmyelinated nerve fibers. Autonomic, sensory and motor nerves are affected, and each category can contribute to the development of the characteristic neuropathic diabetic foot ulcer (Fig 1).

Since these ulcers are highly pathologic and notoriously difficult to treat, the first priority for the clinician is to prevent them from occurring. For the most part, the burden of prophylaxis lies with the primary care physician, and the first rule is to appreciate that **every** diabetic patient is at exaggerated risk of neuropathy and resultant foot problems.

Risk increases significantly when the patient is a smoker, over 40, hypertensive, obese, black, Hispanic or Native American. Unfortunately, it is not uncommon to find all of these factors combined in a diabetic patient.

Figure 1*



*Adapted from Levin and O'Neal.[1]

Charcot foot

The anatomy of wounding

Anatomy plays a major role in the distribution as well as the development of diabetic foot ulcers. Essentially, the majority are pressure wounds in a hypersusceptible host.

The healthy foot bears the pressure of walking over 100,000 miles in a lifetime. Each step applies 3.59 kg/cm² over the first metatarsophalangeal joint and 5.44 kg/cm² over the hallux and 6.21 kg/cm² over the second metatarsophalangeal joint (Fig 2).[9]

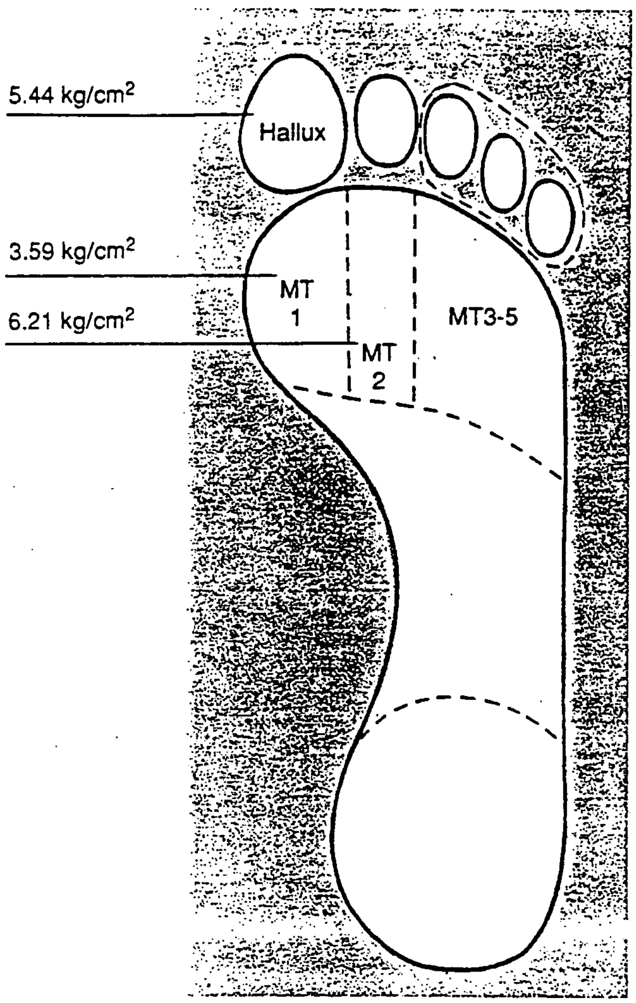
These pedal pressures tend to be increased in diabetic patients with neuropathy, and increase even more in those with ulceration. Most importantly, once the foot becomes insensate, the patient is unaware of what is happening.

Not unexpectedly, the typical neuropathic diabetic foot ulcer occurs on the plantar surface of the foot in areas of maximum pressure.

In the absence of acute trauma, these ulcers are usually preceded by erythema, blisters and/or calluses over the affected areas. The skin hypertrophy that produces the callus is a bodily defense mechanism, but it is also an abnormality that leads to crushing of the capillaries and tissue beneath it and subsequent necrosis and ulceration. Therefore, a callus on a diabetic foot should be treated as a precursor to an ulcer, and probably indicative of abnormal pressure and probably diminished sensation.

Anatomic deformities of the foot are common accompaniments of diabetic neuropathy. The most frequent deformity is cocked up toes (claw toe, hammer toe), and the most profound deformity is the so-called "Charcot foot." This osteoarthropathy originates with failure of the sympathetic nerves that constrict blood vessels supplying the foot. The unregulated vessels dilate, causing increased circulation. Pedal pulses are strong and bounding and the foot becomes edematous, erythematous and hot to the touch. The condition is often mistaken for an acute infection, although the WBC is usually normal.

Figure 2 **Walking pressure on the human foot**



(X) capillary refill

Examining the diabetic patient's feet

With all mature diabetic patients, one should proceed on the assumption that neuropathy is present until proved otherwise and that the feet are at risk even if the patient makes no complaint and the feet appear intact.

As noted previously, while examination of the feet may be bypassed in four out of five diabetic patients, no examination should be considered complete unless it is specifically included. The patient should remove shoes and socks, men should roll up trousers or remove them and women should remove pantyhose.

The limbs should be inspected for signs of vascular insufficiency (Table 1) as well as neuropathy. These signs include turgor, discoloration, a shiny avascular atrophic appearance and loss of hair on the toes. Touching the feet can help identify changes in skin texture and temperature.

Delayed venous or capillary filling time can be measured simply by having the patient lie supine with feet elevated to a 45-degree angle until one or both feet blanch and then sit upright with the feet dependent until the color returns to normal. This should occur within 15 seconds or less. A filling time of 15 to 25 seconds indicates moderate ischemia; above that, the ischemia is severe.

One should examine the feet carefully for general appearance and hygiene as well as evidence of ulceration or its precursors. Risk areas include hot-spots, boney prominences, corns, calluses, blisters, ingrown toenails and atrophied fat pads.

Shoes and stockings should also be inspected. Neuropathy as well as vanity can cause the diabetic patient to choose too-small of a shoe size or hosiery with seams. Additionally, the inside of the shoe may contain projections, foreign bodies or even exposed nailheads that go undetected if the foot is insensate.

Table 1 **Signs and Symptoms of Vascular Disease in the Lower Extremity***

1. Intermittent claudication
2. Cold feet
3. Nocturnal pain
4. Rest pain
5. Nocturnal and rest pain relieved with dependency
6. Absent pulses
7. Blanching on elevation
8. Delayed venous filling after elevation
9. Dependent rubor
10. Atrophy of subcutaneous fatty tissue
11. Shiny appearance of skin
12. Loss of hair on foot and toes
13. Thickened nails, often with fungal infection
14. Gangrene

*Adapted from Levin and O'Neal.[1]

Assessing diabetic neuropathy

The testing needed to evaluate diabetic neuropathy of the foot is relatively easy to perform and does not require specialized equipment. The "instrumentation" is illustrated in Fig 3. It consists of a 128 tuning fork, a calibrated monofilament plastic probe, a Q-tip and the physician's fingers. The sensations to be evaluated are vibration, pressure, touch and proprioception. Temperature sense is included by some.

The procedure is as follows:

Vibration—Strike a 128-cycle tuning fork and touch it to each toe and other areas such as the malleoli. In the absence of neuropathy, vibration will be perceived for not less than 15 seconds, and usually for 20 to 25. At each test, the examiner can apply the tuning fork to the same area of his or her own foot to compare the vibratory perception. If the patient's perception is diminished or absent at the extremities, the fork should be moved proximally and tried again to produce a semi-quantitative measure of sensation deficiency.

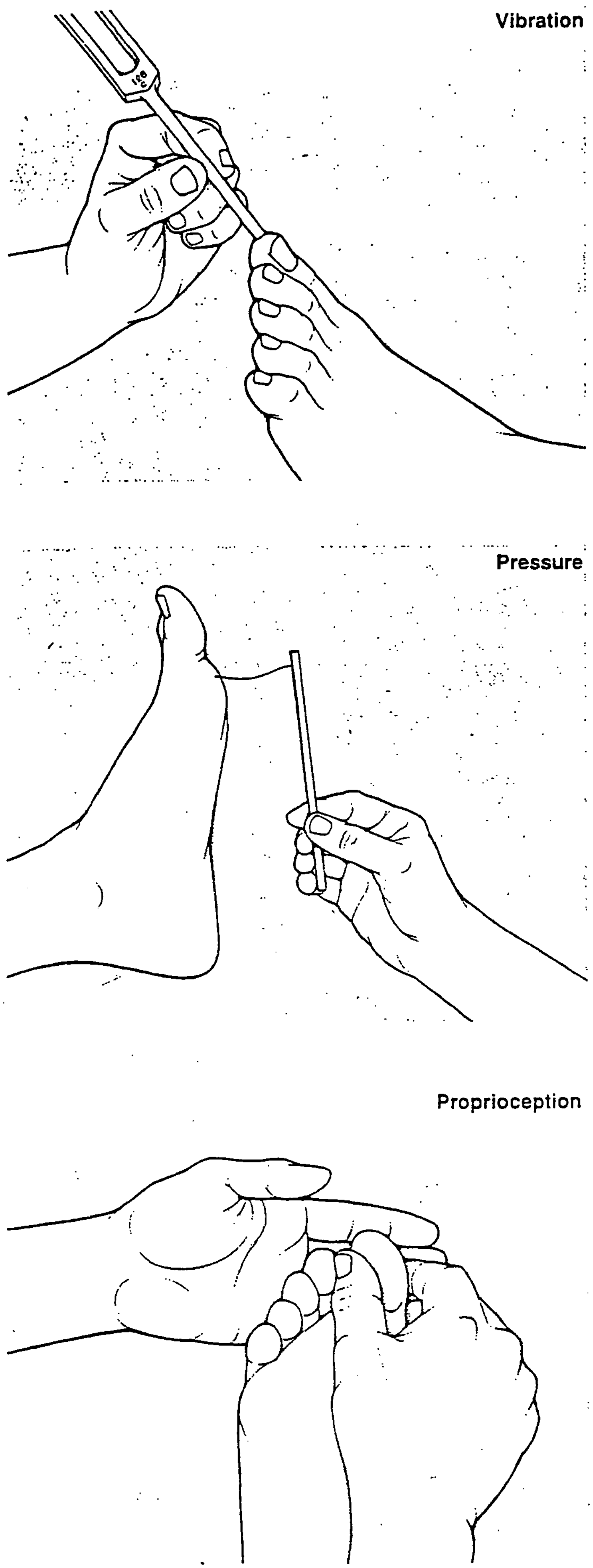
Pressure—The test instrument is a Semmes Weinstein monofilament nylon probe calibrated to a thickness of 5.07. These probes can be obtained from the Gillis W. Long Hansen's Disease Center, Carville, Louisiana at a minimal cost. Other thicknesses are available but rarely necessary. The probe is simply pressed against the skin until it buckles, at which time the patient should be able to detect its presence. Different areas of the sole of the foot should be tested and the patient should be able to identify which area is being touched. Buckling of the 5.07 monofilament occurs at 10 grams of linear pressure and is the limit used to detect protective sensations. If the patient does not detect the probe, significant neuropathy is present and the patient is at significant risk for injury.

Touch (sharp and blunt)—This simple modification of the traditional pinprick test is performed with a wooden Q-tip snapped off to provide a sharp point to test for sharp perception. The cotton end is used to evaluate sensitivity to blunt touch. The tests should be performed out of the patient's line of sight and, like the others, on several different areas starting distally and moving successively more proximally.

Proprioception—For this test, the physician simply cradles the foot, holds the toes gently by their sides and out of the patient's sight and moves them up and down. With significant neuropathy, the patient will be unable to determine their position in space when asked.

Together with the patellar and achilles reflexes, these simple tests provide a practical index of peripheral neuropathy. While loss of sensation varies from patient to patient, vibratory perception is the first to be lost, followed by the achilles reflex and, finally, perception of pain and touch.

Figure 3



If an ulcer is present

Not every wound in a diabetic's foot is a neuropathic diabetic foot ulcer. Nevertheless, those that are share characteristics that, if not definitive, are at least highly suggestive of the diagnosis and lend themselves to aphoristic summation:

If it doesn't hurt, it's neuropathic.
Alcoholics and patients with Hansen's disease may develop painless ulcers, but in the person with diabetes, diabetic neuropathy is the probable culprit. On the other hand, a painful ulcer in a diabetic patient may still have a neuropathic component coupled with pain due to ischemia or necrotizing infection.

If it's dorsal, it's probably traumatic.
As previously noted, diabetic neuropathic ulcers have a strong predilection for pressure areas on the plantar surface of the foot and/or toes. Anatomic deformities may revise the pressure points, but rarely to the dorsum. Foreign bodies or tight shoes may inflict wounds with equal pathology and progression but, once cured, they are less likely to recur since the cause of trauma can be more easily identified and removed.

If it doesn't heal in a month, it's likely to be deeply infected or ischemic or both.
Most neuropathic diabetic ulcers are infected and seemingly superficial wounds are often like the cap of a volcano, masking seething layers of infected, necrotic tissue below. Without aggressive debridement, the true extent of the wound is obscured, the character of infection, especially underlying osteomyelitis, is unappreciated and the wound simply won't heal. Superficial granulation may occur, but as soon as pressure is reapplied (as in ambulation), it breaks down. The refractory wound is virtually the model of the neuropathic diabetic ulcer. Keep in mind that wounds or ulcers not responding to aggressive treatment or that occur in atypical areas not explained by trauma should be considered for biopsy to rule out possible malignancy. If ischemia is suspected, refer for vascular evaluation.

If the ulcer probes to bone, there's probably osteomyelitis.
Beneath the surface of the typical diabetic neuropathic ulcer, infection spreads insidiously to invade surrounding subcutaneous soft tissue and tendons. The wound flora is commonly polymicrobial and resistant to the usual oral antibiotics. Once it reaches bone, invasion is the rule rather than the exception.

If it's gangrenous, it's lost.
This final aphorism is—fortunately—FALSE. With expert, intensive care, especially with revascularization, aggressive debridement and topical application of growth factors, better than 80% of extremities and limbs with evidence of gangrene can be salvaged if treatment is applied in time. The necessary level of care, however, is a comprehensive wound management team effort.

This brief list of truisms, which are probable, underscores the need for a systematic approach to categorizing the risk that diabetic neuropathy imposes for the patient's foot, pathogenicity of the ensuing wounds and, ultimately, a practical triage for managing them effectively.

Categorizing the neuropathic foot and ulcer

A simple method of grading risk level for the diabetic foot divides it into four categories based on neuropathy and history of ulceration (Table 2).

Under this system, those in category 3 will almost surely develop an ulcer while those in categories 2 and 1 have decreasing risk of developing one.

If an ulcer is present, its severity may be categorized by a Wound Grading System (Table 3). Risk for the still-intact diabetic foot and wound grade for those that become ulcerated establish the criteria for wound management. For a patient in risk category 0, for example, the goal is wound prevention, and

Table 2 **Foot Risk Category**

Category	Semmes Weinstein protective threshold (5.07)	Prior neuropathic ulcer	Ulcer associated with foot deformity
0	Intact	No	No
1	Lost	No	No
2	Lost	Yes	No
3	Lost	Yes	Yes

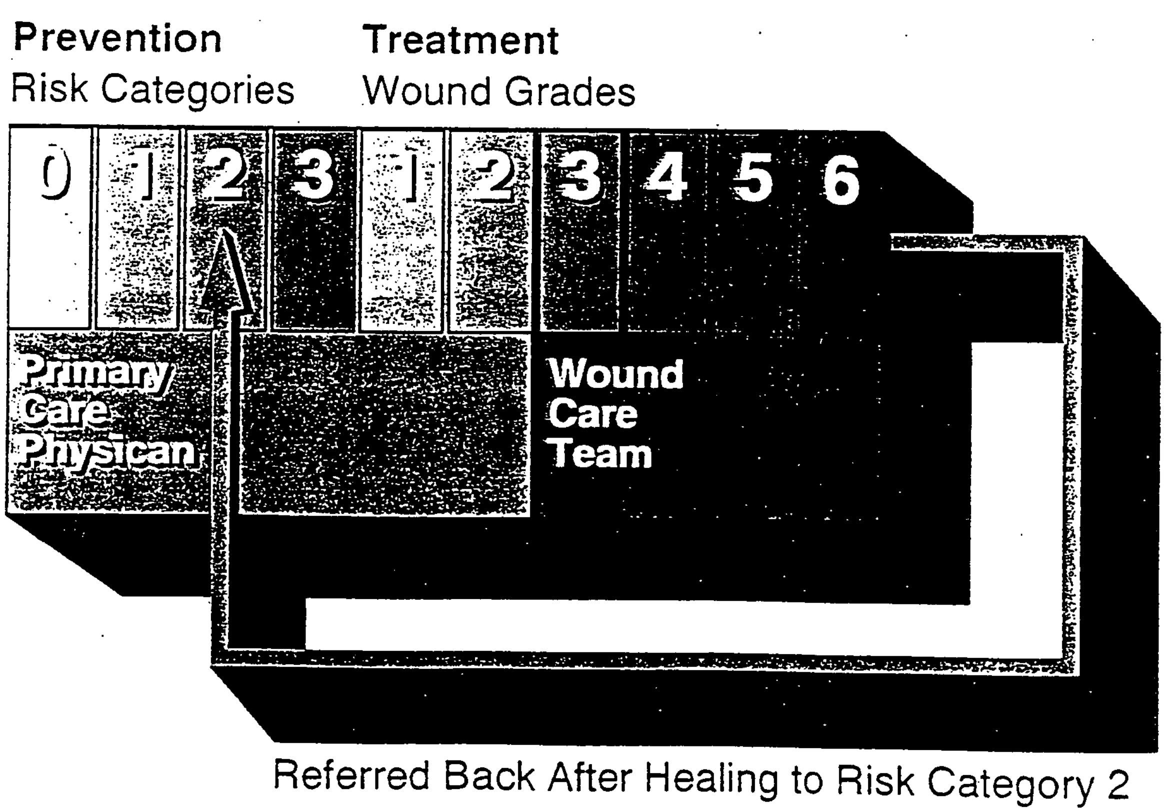
Note: If patient has signs of peripheral vascular disease they should be moved to the next higher category.

Table 3 **Wound Grade***

Grade	Thickness of ulcer	Involvement of tendon, bone, ligament, joint	Infection, necrosis, gangrene
1	Partial-Dermis and Epidermis	None	Minor or None
2	Full-Subcutaneous Only	None	Minor or None
3	Full	Yes	Minor or None
4	Full	Yes	Abscess and/or Osteomyelitis
5	Full	Yes	Necrotic Tissue in Wound
6	Full	Yes	Gangrene in Wound and in Surrounding Tissue

*Wound Care Center® protocol.

Figure 4 **Clinical Spectrum**



[illegible] patient education [illegible] and observation [illegible] other end of the [illegible] with a full thickness [illegible] gangrenous ulcer, the objective is to save the patient's limb and, perhaps, his or her life. These patients need to be hospitalized and their care require [illegible] capabilities of a com[illegible] wound care team [illegible] include the primary [illegible] team, endocrinologists, infectious disease specialists, vascular surgeons, plastic surgeons, general surgeons, orthopedists, podiatrists, specialized wound [illegible] nurses, physiotherapists, [illegible], and social workers.

[illegible] evaluation of the patient [illegible] the wound initiates [illegible] triage for how it should be managed and by whom. As a practical matter, diabetic patients with neuropathy but still-intact skin, excepting those with foot deformities, are best managed by the primary care physi[illegible]. Those with grade 3 to 6 ul[illegible] are best referred to a specialized wound care team (Fig 4). The borderline occurs with the grade 2 ulcer, full thickness, but involving only subcutaneous tissue.

The key question is whether one can be sure the wound is grade 2. Surface appear[illegible] and superficial debridement can be dangerously misleading. Generally, if bone is visible or palpable in a wound, the patient should be referred.

The apparently small neuropathic ulcer shown in Fig [illegible] illustrates the problem. Its [illegible] belies the true depth and ex[illegible] necrosis revealed by the [illegible] debridement shown in Fig 5B. There is little chance that such an ulcer will heal while infection and necrosis proceed beneath the surface.

In this patient, the de[illegible] arch accounts for the [illegible] location and is an indi[illegible] that the patient's risk [illegible] be upgraded to category [illegible]. With a similar ulcer in an anatomically [illegible] mal foot, the primary care physician might initiate treatment empirically, bearing in mind the aphorism that if it doesn't heal in a month, the infection is deeper and more severe than it appears and it may require biopsy, and should be referred.

Care for the foot at risk

It cannot be overemphasized that the feet are at risk for any person with diabetes regardless of insulin dependency or current evidence of neuropathy. No diabetic patient should smoke. All require special precautions concerning nutrition, metabolism, weight control and avoidance of environmental hazards ranging from infection to weather extremes and household obstacles. In particular, patients' feet should be examined routinely without waiting for sensory loss, preferably three or four times a year and more often if there is an evident foot problem. The examination can be carried out by the physician or other health care professional.

The Charcot foot is an extreme risk. At the acute onset, it is characterized by a foot that is warm, edematous and erythematous, and with bounding, pulsing and prominent veins. The initial treatment is to keep the foot from weight bearing. If this is not done promptly, stress fractures of the bones of the foot and ankle may occur. The arch collapses, causing a "rocker bottom" appearance and a redistribution of walking pressure to the plantar surface of the arch. If proper shoes and weight redistribution are not accomplished, ulceration will develop.

Sensory neuropathy is the rule in patients with this condition and, without intensive expert care and special shoes, the Charcot foot will almost always be subject to ulceration. Thus,

Figure 5A*

Figure 5B*

*Adapted from Levin and O'Neal.[1]

No weight for Charcot foot.

even if the foot appears intact upon examination, the patient is best referred without delay to a facility that specializes in this complication.

Education to keep the neuropathic foot intact

Once sensation loss is detectable, the patient advances to risk category 1, and should be seen more frequently (ie, every 3 months) and taught to cooperate in specific prophylactic measures. These include daily inspection, foot hygiene and probably the addition of professionally designed soft insoles to any commercial footwear.

Education is essential since patients must appreciate the rationale for the risk-reduction rules in which they are expected to comply. Written guidelines should be provided to supplement verbal instructions.

A basic menu of foot care guidelines for patients is summarized in this monograph. Duplicate them for distribution to your patients. Education on good foot care practices is especially important for people with diabetes, and particularly those who have had the disease longer than 20 years, or patients over 40 years of age.

It should be emphasized that the guidelines are only preventive, and apply to all patients who still have adequate protective sensation, no foot deformity, and no history of plantar ulceration.

Patients who have had previous neuropathic ulcers, and particularly those associated with foot deformities should be referred to a pedorthist or podiatrist for custom orthotic devices and prescription footwear.

When prevention fails

If the ideal is to prevent diabetic ulcers while the foot is still intact, the reality is that the typical diabetic ulcer is already present when the foot is first examined. Neuropathy delays the complaint well beyond the threshold that would drive an unaffected patient to treatment.

Nevertheless, differential diagnosis and evaluation is still needed before treating any newly-discovered foot ulcer:

- Is it a diabetic ulcer?
- Is it neuropathic?
- Is it infected?
- Is it traumatic?
- Is it malignant?
- Is there vascular involvement?
- What is the wound grade?
- Is it better to treat or to refer?

Assuming the patient is known to have diabetes and the foot is neuropathic, infection should be presumed in any plantar ulcer, particularly if it has persisted for over a month.

As noted earlier, the extent of the wound will probably exceed its superficial appearance. Thus, the initial evaluative step is to **x-ray, debride and culture.**

Debridement should be aggressive and thorough. One should use a curette and explore the depth and breadth of the wound. Necrotic and infected tissue should be curetted or removed until clean, healthy tissue is exposed. If necrosis extends to bone, **osteomyelitis should be presumed unless proven absent.** Curettings or a bone biopsy should be examined and cultured.

Culturing of the wound should include aerobes and anaerobes. Fungus cultures should be considered in special situations. Separate collection tubes will be required for each category and should be on hand in advance. Material for culturing should come from deep tissue curettings rather than superficial swabs. Polymicrobial infection should be anticipated, and the laboratory should be instructed not to discard specimens as "contaminated." Sensitivities should be obtained **for all microorganisms identified.**

If there is significant vascular involvement, it will probably be manifested in the physical appearance of the limb and/or pain beyond the perimeter of the ulcer site. If it is suspected, a thorough vascular evaluation is indicated.

For wounds amenable to treatment by the individual physician (usually most grade 1 and some grade 2), four points dominate the therapeutic protocol:

1. off-weighting
2. control of infection
3. appropriate wound dressing
4. acute observation including vascular studies.

Off-weighting. No patient with a diabetic neuropathic ulcer can afford to put pressure on the wound until it is **totally** healed. The patient should be provided with crutches, walker, wheelchair, contact cast, or special healing sandals that unweight the wound area and instructed **never** to put weight on the ulcerated foot without explicit permission.

As long as the wound is spared full pressure, limited ambulation may be allowed once there is 100% epithelialization with the skin maturing nicely and minimal or no drainage. However, a protective dressing should remain in place and the patient should wear only protective shoes.

Infection control. It is important to initiate antibiotic treatment while awaiting the results of cultures and sensitivity testing. However, one should be prepared to revise treatment promptly when the culture results become available. Frequently, two or more antibiotics in combination, and possibly an antifungal as well, will be required. Emphasis should be on **bactericidal** rather than merely bacteriostatic agents.

If oral antibiotics are used, patients should be carefully instructed to telephone at once if they experience signs of increasing infection such as increases in pain, discomfort or increasing erythema, or evidence of lymphangitis or increased drainage.

Parenteral antibiotics are indicated for patients who are septic or have significant cellulitis or leukocytosis. These patients should be hospitalized at once.

Topical antibiotics and silver sulfadiazine can help protect the surface of infected wounds, but they should never be considered as the primary or only therapy. In addition, many widely used topical antiseptics should be expressly avoided. Specifically:

Preparations containing povidone-iodine, hydrogen peroxide, acetic or other acids should not be used.

These agents inhibit fibroblast formation, are cytotoxic, and can inhibit granulation tissue formation, and impair wound healing.

Dressing the wound. Specific wound dressings have had vehement advocates and detractors for years. In general, however, wound care specialists acknowledge that the moist environment provided by occlusive dressings is favorable to surface healing but should be avoided in deep wounds. Wet-to-dry dressings after initial debridement are preferred for most superficial wounds.

Observation. Wound healing is a dynamic process, and if healing is not progressing, it is probably retrogressing. Frequent observation is needed to determine which direction a diabetic ulcer is taking, and progression should be measured against standardized criteria. A simple Functional Assessment (FA) grading utilized by Wound Care Centers® provides a convenient guide (Table 4).

By these criteria, a treated wound that has not advanced at least one functional assessment in a month is highly suspect, and if it has not achieved a FA of 3 or 4 within two months, it should probably be referred to a specialized wound care team (Table 5).

The basic reason for establishing healing deadlines for neuropathic diabetic ulcers is that the foot as well as the patient is already compromised and at risk beyond what is apparent in the wound itself. Unpredictably, and within days or even hours, the seemingly indolent ulcer can convert to a fulminating systemic infection and part or all of the foot may be beyond salvage. In effect, no wound in a neuropathic diabetic foot is trivial. All should be observed professionally at intervals of no more than a week, with the patient warned to telephone **immediately** if signs of systemic infection (including malaise and lethargy as well as fever or those previously mentioned) occur.

When wounds won't heal

The four most common reasons for non-healing of neuropathic diabetic ulcers are:

1. failure to "off-weight" the foot
2. unappreciated deep infection (including osteomyelitis)
3. vascular compromise
4. noncompliance.

Noncompliance includes failure by the patient to keep pressure off the ulcer site, failure to take medication or keep the wound dressed and failure to keep the underlying disease under control.

Hyperglycemia, poor nutrition and poor hygiene all retard the healing process. So, too, does vascular insufficiency, which lessens the ability of antibiotics, oxygen and other nutrients to be delivered to the wound. However, the principal obstacles in most cases are continuing pressure and continuing infection. Individually or in combination they convert the grade 1 wound to grade 2 and, ultimately, grade 2 wounds to grades 3 or worse.

It is impractical for most individual physicians to manage the non-healing grade 2 wound or those which have progressed beyond it. Regardless of the reasons for progression, it will require a comprehensive team effort to arrest it. It follows that, while the foot is still salvageable, the help of an experienced wound care team should be enlisted.

Table 4 **Functional Assessment (FA) of Wound Healing***

	Epithelialization	Skin maturity	Drainage	Dressing needed
	< 100%	No	Yes	Yes
2	100%	No	Yes	Yes
3	100%	Maturing	Minimal	Protective
4	100%	Mature	No	No

*Wound Care Center® protocol.

Table 5 **Criteria for Referral to a Wound Care Team**

- Wounds of grade 3 or higher
- Grade 2 wounds that fail to show significant improvement within a month
- Wounds that fail to heal in two months
- Wounds in feet that are dysvascular as well as insensate
- Wounds that repeatedly break down
- Any wound (or any patient) with evidence of Charcot foot

Wound Care Centers®

Most primary care physicians work closely with diabetologists, orthopedists, podiatrists, infectious disease specialists, vascular surgeons, and other related specialists in caring for their patients with neuropathic foot ulcers. The extension of what may be a loose association into formally organized and coordinated Wound Care Centers is a recent but logical development.

Much of the protocol of the Wound Care Center recapitulates that followed by the primary care physician. The difference lies in the additional breadth and depth of assessment that the capacities of a complete wound care team permit, and the treatment options that can be marshaled once the extent of the wound has been established.

The typical Wound Care Center algorithm for managing refractory wounds includes the following components as needed:

- Assessment
- Vascular studies
- Revascularization
- Infection control
- Debridement
- Growth Factor Therapy
- Protection
- Referral
- Skin grafting
- Prevention

In each category, the team approach reinforces the contribution of individual members. For example, if radiographic studies uncover early signs of Charcot's arthropathy, customized protective footwear can be ordered and corrective exercise protocols can be instituted without delay.

At Wound Care Centers, patients participate in a comprehensive program on an outpatient basis which includes, when indicated, the use of newly developed growth factor technology. The program is conducted by an integrated multidisciplinary team headed by physicians and nurses especially trained in the use of management protocols and the growth factors, and including non-invasive vascular technicians, orthotists, pedorthists, nutritionists, physical therapists and support staff.

Treatment is preceded by an extensive assessment which includes history, physical examination and quantitative measurement and grading of the wound. Data is maintained in a database for objective evaluation of healing outcome.

Where peripheral vascular disease is suspected, transcutaneous oxygen measurements (T_CPo_2) are made to determine tissue perfusion in the lower leg and doppler waveform analysis to determine the potential level of occlusion. Angiographic studies usually follow if the T_CPo_2 is below 30 mm Hg.

Vascular reconstruction is recommended if the angiograms confirm an occlusive process and the patient and his/her arteries are amenable to reconstruction.

In nearly all cases, aggressive wound debridement is conducted, including excising bone if osteomyelitis is suspected.

A particular feature of the Wound Care Centers is the introduction of biological growth factor technology to supplement standard wound management.

This innovative technology is based on the discovery of multiple growth-promoting factors within the platelet, liberated as part of the clotting cascade.

The functions of five of these factors have been identified and their role in the formation of wound granulation tissue, capillaries, and epithelium defined (Table 6).[10]

Table 6 **Growth Factor Actions***

Growth Factor	Activity	Cell Interaction	Function
PF-4	Chemoattractant	Neutrophilis Monocytes	Clears debris and bacteria
PDGF	Mitogen Chemoattractant	Fibroblasts Smooth muscle cells	Produces extracellular matrix
TGF-ß	Chemoattractant Increases matrix synthesis Stops division	Monocytes Fibroblasts	Infection control and strengthens matrix
PDAF	Chemoattractant	Endothelial cells	Restores vascular system
PDEGF	Mitogen Chemoattractant	Epidermal cells	Produces epidermal skin

*Adapted from Fylling.[10]

[illegible] the patient's own blood, [illegible] autologous preparation [illegible] derived wound healing factors (PDWHF) is prepared by special facilities in the Wound Care Center[8] and then applied, in solution, directly to the wound. The growth factors interact at the cell receptor sites in the wound area, causing new [illegible]

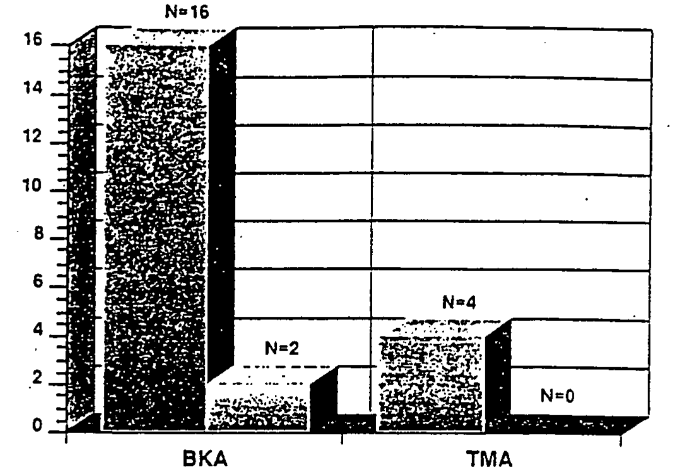
[illegible] conjunction with comprehensive wound management, PDWHF has produced often remarkable results, including a shortening of wound-healing time and more complete wound healing (Figs 6 & 7).[11]

At present, facilities for preparing and administering PDWHF are limited to specialized Wound Care Centers. Although [illegible]echnology is new, numerous studies, including double blind comparisons, have confirmed its contribution to the wound healing process.[10,12-14]

The most striking result of appropriately applied PDWHF technology has been more rapid and complete healing of diabetic foot wounds and the salvage of limbs that conventional therapy would have consigned to amputation (Fig 8).

The primary care physician maintains the care of the underlying diseases while the Wound Care Center treats the wound. Once the wound is healed, the patient is referred back to his/her primary care physician.

Figure 8 Limb Salvage Study*



*Adapted from Knighton DR et al.[13]

ight Plantar Foot 45 Year Old Diabetic Male*

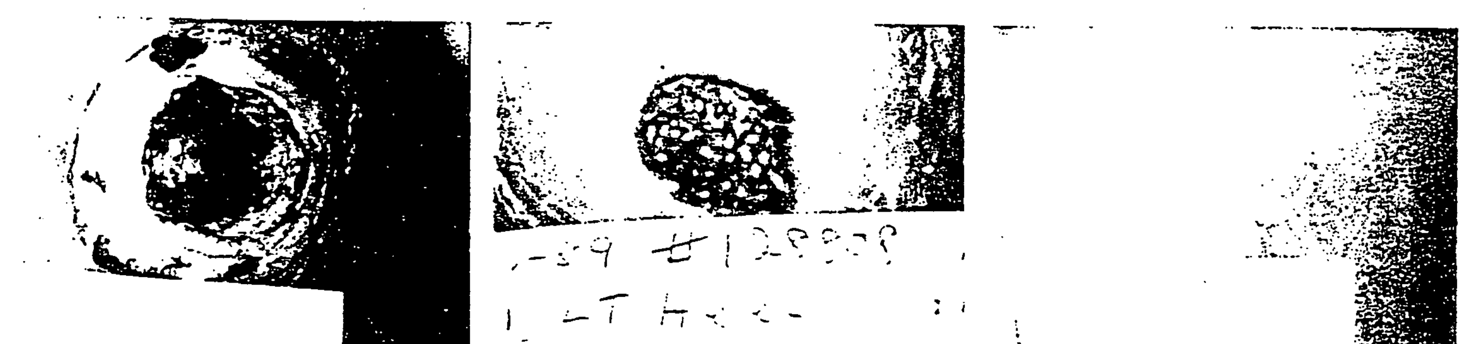
1st Visit

Week 6

Week 11

*Adapted from Poucher et al.[11]

Figure 7 Left Plantar Heel 43 Year Old Diabetic Female*



After Debridement

Week 8

Week 12

*Adapted from Poucher et al.[11]

Neuropathic ulcers of the foot constitute a major hazard for the diabetic patient which is often overlooked until it is too late for prevention. Early and thorough examination of the feet of all patients with diabetes and education of the patient to appreciate the risk and maintain good hygiene are the essential steps toward prophylactic care.

If the foot becomes neuropathic, the risk is magnified, and when an ulcer is detected, its severity should not be underestimated.

While many neuropathic diabetic ulcers can be managed effectively by the primary care physician, grade 3 to 6 wounds and any treated wound that has not improved within a month and/or healed at two months should be regarded as deeply infected and will usually require management by a specialized wound care team.

The team approach to management of the neuropathic diabetic foot, utilizing new techniques including growth factors, has produced an impressive record of success, including salvage of severely compromised limbs and rehabilitation of many patients who would otherwise be permanently disabled.

References:

1. Levin ME, O'Neal LW, eds. *The Diabetic Foot*. 4th ed. St. Louis, Mo: CV Mosby Co; 1988;ix. 2. Palumbo PJ, Melton LJ III. Peripheral vascular disease and diabetes. In: *Diabetes in America: Diabetes Data Compiled in 1984*. Washington, DC: US Govt Printing Office; 1985: Chap XV. NIH publication 85-1468. 3. Levin ME: 1991, Personal Communication. 4. Ebskov B, Josephsen P. Incidence of reamputation and death after gangrene of the lower extremity. *Prosthet Orthot Int*. 1980;4:77. 5. Drury TF, Danchik KM, Harris MI. Sociodemographic characteristics of adult diabetes. In: Harris MI, Hammer RF, eds. *Diabetes in America*. Bethesda, Md: 1985. NIH publication 85-1468. 6. O'Brien IAD, Corrall RJM. Epidemiology of adult diabetes and its complications. *N Eng J Med*. 1988;318:1619. 7. Bailey TS, Yu HM, Rayfield EJ. Patterns of foot examination in a diabetes clinic. *Am J Med*. 1985;78:371. 8. Jauw-Tjen L, Brown AL, Jr. Normal structure of the vascular system and general reactive changes of the arteries. In: Fairbairn JF, Juergens JL, Spittell JA, eds. *Peripheral vascular disease*. Phila, Pa: WB Saunders Co; 1972. 9. Sanders LJ: 1989, Personal Communication. 10. Fylling CP. Comprehensive wound management with topical growth factors. *Ostomy Wound Manage*. 1989;22:62. 11. Poucher RL, Leahy JD, Howells G. Case report: active healing of diabetic wounds utilizing growth factor therapy. *Wounds* 1991;3:66. 12. Doucette MM, Fylling C, Knighton DR. Amputation prevention in a high-risk population through comprehensive wound-healing protocol. *Arch Phys Med Rehabil*. 1989;70:780. 13. Knighton DR, Fylling CP, Doucette MM. Wound healing and amputation in a high risk diabetic population. *Wounds*. 1989;1:107. 14. Knighton DR, Fylling CP, Fiegel VD, Cerra F. Amputation prevention in an independently reviewed at-risk diabetic population using a comprehensive wound care protocol. *Am J Surg*. 1990;160:466.

PATIENT CARE GUIDELINES

GENERAL CARE AND HYGIENE

- **Never go barefoot either indoors or out.**
- **Inspect your feet daily.** Use a mirror and pay particular attention to soles and between toes. (If you have difficulty in doing this, get help from a family member.)
- **Wash your feet daily.** Test the water first with your hand or elbow to be sure it's not too hot.
- **If your feet become excessively dry, lubricate them.** (Spread a thin film of moisturizing cream on the soles while still wet immediately after bathing. Take care **not** to get the cream in between your toes.)
- **Don't use garters or elastics to hold up stockings.**
- **Don't use panty girdles that are tight around the legs.**
- **Avoid exposing your feet to extremes.**
  —Do not walk on hot sand or pavement in summer.
  —Protect feet against sunburn with a sunscreen.
  —Check temperature of bath water before bathing.
  —Never use hot water bottles or heating pads to warm cold feet (wear socks at night in bed if feet feel cold).
  —Beware of car heaters on long trips.
  —Keep nails trim and cut straight across (don't round corners).
  —Never cut corns or calluses yourself. (However with careful instruction from your physician, you can be taught how to work on calluses using pumice stones or emery boards if they aren't too thick.)
  —Never use commercial corn or callus removers, foot pads or arch supporters.
  —Don't use adhesive tape on your feet.
  —Don't "ice down" your feet if they feel hot.
  —Never use hot or cold soaks for your feet.
  —Keep toes clean and free of debris between them.
- **Be sure to see your doctor or podiatrist for a foot inspection at least twice every year.** Be sure they know you have diabetes.
- **Notify your doctor or podiatrist promptly if you develop a blister, puncture or sore on your foot.**

FOOTWEAR

SHOES

- **Buy only comfortable, well-fitting shoes.** Have the clerk fit them for you, walk around in them and be sure they are comfortable **immediately**.
- **Buy new shoes late in the day.** Feet enlarge slightly during the day and shoes that fit in the morning may be too tight by noon.
- **Choose shoes with soft leather uppers** that can mold to the shape of your feet (modern walking or running shoes may be beneficial).
- **Never buy shoes with open toes or heels.**
- **Never wear or buy sandals,** particularly those with thongs between the toes.
- **Have your doctor or podiatrist inspect new shoes** to be sure of proper fit and construction.
- **Never wear new shoes more than 2 hours at a time.**
- **Don't wear any shoes more than 5 hours at a time.** You should have one pair for morning, one for the afternoon and one for evenings around the house.
- **Never wear shoes without socks or stockings.**
- **Always inspect the inside of your shoes carefully before putting them on and after taking them off.**

HOSIERY

- **Never wear socks or stockings of 100% synthetic material.** Proper materials include 100% cotton and blends. Wool should not be worn except in winter, and then only if you are sure it is non-irritating. (Check with your physician or podiatrist for socks or stockings made specifically for diabetics.)
- **Never wear socks or stockings with seams.**
- **Never wear socks or stockings with holes or mends.**
- **Wear only clean socks, and change them daily.**
- **Inspect socks or stockings carefully before and, particularly after, wearing them.**